ADVANCES IN GERD

Current Developments in the Management of Acid-Related GI Disorders

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Hiatal Hernia and the Treatment of Acid-Related Disorders

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G&H Could you describe the varying manifestations of hiatal hernia?

JP Hiatal hernia is, in general terms, a herniation of any element of the abdominal cavity through the esophageal hiatus of the diaphragm.

Hiatal hernias can be classified into four types. Type 1 is the classic "sliding" hiatal hernia, where a widening of the hiatal canal allows part of the stomach to slip into the chest. This is a very common phenomenon that has been shown in prevalence studies to occur in anywhere from 10% to 80% of the adult population. It represents a simple laxity of the esophageal gastric junction.

The other types of hernia occur when Type 1 progresses. A Type 2 hernia results from, once again, a defect in the gastroesophageal junction and the crura, but the gastroesophageal junction remains fixed at the crural diaphragm and a portion of the stomach slips to the side of the esophagus. This has the potential to progress to a nearly complete herniation, or upside-down stomach. Type 3 hernias are a combination of types 1 and 2, where a progressively larger hernia forms through the hiatus and, essentially, the gastroesophageal junction and proximal stomach both migrate into the chest. Type 4 hiatal hernia results from the same type of progressively larger hiatal defect, but other organs such as the spleen, the small intestine, and even the colon can actually herniate, along with the gastroesophageal junction, into the chest.

G&H What causes the occurrence of hiatal hernia?

JP Whether hiatal hernia is a congenital condition or due to some form of trauma is somewhat controversial. We believe that most Type 1, Type 2, and smaller Type 3 hernias are likely due to aging and the normal wear and tear and progressive laxity of the musculature. However, we do see some younger patients with these problems, which would suggest a congenital component. There is also a belief that other stressors, including the intra-abdominal pressure that occurs with pregnancy and the strain from exercise or heavy lifting, can cause a herniation. In addition, there are traumatic herniations, from car accidents or falls, for example, where the pressure from the abdominal traumatic event pushes the abdominal contents into the chest and disrupts the hiatus orifice.

G&H Are there any groups in the general population where hiatal hernia is particularly prevalent?

JP Obese people are certainly more prone to the development of hiatus hernia. In fact, our group recently published a study in *Gastroenterology* that showed a correlation between waist circumference and axial separation of the crura diaphragm and the lower esophageal sphincter, which means that the lower esophageal sphincter is moving into the chest.

There is no well-defined split between genders but, as stated above, pregnancy can certainly exacerbate this condition. Some studies suggest a transient herniation of the esophageal gastric junction during pregnancy that, in many women, returns to normal after the baby is delivered.

G&H How does the presence of hiatal hernia affect the development of reflux disease?

JP The esophagogastric junction is the antireflux barrier and the primary determinant of reflux potential. It is made up of a number of different physiologic and anatomic components, including the lower esophageal sphincter, the crural diaphragm (the sling muscles that wrap around the esophageal gastric junction), the phrenoesophageal ligament, gastric sling fibers, and the conformational change that occurs, known as the gastroesophageal flap valve. The propensity to reflux is based on obliteration or alteration of all of these components. If a patient has a large hiatal hernia but the lower esophageal sphincter muscle is functioning well, he or she will most likely not develop reflux disease. In order for reflux to occur, multiple defects in the antireflux barrier must be present and the two main sphincters, the crural diaphragm, or hiatus, and the lower esophageal sphincter, must both be incompetent or relaxed. This is the reason why healthy individuals, when swallowing, do not experience reflux. The crural diaphragm is still contracting and pushing the stomach contents down, whereas during a transient lower esophageal sphincter relaxation or belch reflex, both the diaphragmatic crura and the lower esophageal sphincter relax, allowing air passage.

Thus, many people who have a hiatal hernia do not actually have reflux, because the lower esophageal sphincter is working well and protecting them. Our group has also recently studied the contribution of the crural diaphragm in creating a high-pressure zone. We found that a better predictor of the development of reflux disease was the amount of augmentation that occurred when the diaphragm contracted during inspiration. This measure was shown to be a surrogate marker, signaling an interplay between both the intrinsic lower esophageal sphincter and the extrinsic crural diaphragm.

G&H In patients who do have reflux and GERD symptoms, does the presence of a hiatal hernia change their profile in terms of symptomology and disease severity?

JP The presence of hiatal hernia changes a patient's profile in that patients with more severe forms of gastroesophageal reflux disease (GERD), such as Barrett metaplasia or severe erosive esophagitis, are more likely to have a hiatal hernia. Patients with Barrett esophagus, particularly long-segment Barrett, almost always have a hiatal hernia.

One previous study modeled the relationship between lower esophageal sphincter pressure and the hiatal defect in terms of the axial length. The investigators developed a model of reflux potential that illustrated an interaction where, if lower esophageal sphincter pressure was decreased to below 10 but no hernia was present, it was very unlikely that severe reflux disease would manifest. However, if lower esophageal sphincter pressure was reduced and there was a hiatal hernia, the reflux potential would be significant. In addition to decreasing the protective barrier, hiatal hernia also impedes esophageal clearance after reflux. A hernia creates a pocket of gastric juice that does not clear with swallowing. In addition, any anatomic change that shortens the esophagus, such as hiatal herniation, compromises the peristaltic ability to clear it.

The pathogenesis and progression of reflux disease is determined by both aggressive forces (the causticity of gastric reflux and the number and frequency of reflux events) and defensive forces (protective mucosal tissue and the ability to clear acid). Hiatal hernia affects both the number of reflux events and the ability to clear acid, heightening severity on both fronts.

G&H Does the presence of hiatal hernia affect patients' response to treatment?

JP Response depends on the primary symptom. If the patient's main complaint is of heartburn, it can usually be controlled with proton-pump inhibitor (PPI) therapy. If the primary symptom is regurgitation, PPI therapy falls short. Patients with severe reflux and hiatal hernia who are on high-dose acid suppression will often report that their heartburn is gone but that they can still taste gastric juice in their mouths. These patients, if their quality of life is affected, may need to be sent for surgical treatment of their hiatus hernia.

It is also thought that hiatal hernia patients are more likely to have extraesophageal and pulmonary symptoms, because even if acid levels have been controlled, these patients are still refluxing fluid into the pharynx and possibly the lungs. This is a controversial idea, but it is supported by the knowledge that reflux still occurs in patients on PPIs and that the trachea and larynx do not have the same protective squamous mucosa that lines the esophagus. In addition, I suspect that patients who have a large hernia are more likely to have severe symptoms at night because, once again, they have no protective barrier. They are sleeping, gravity is gone, and they are theoretically spilling gastric juice continually into their esophagus.

G&H What are the current surgical options for repair of hiatal hernia?

JP Antireflux surgery repositions the lower esophageal sphincter into the abdominal cavity and then creates an artificial flap valve, wrapping the proximal stomach around

the esophagus and anchoring it into the abdominal cavity. In addition, it is very important to repair the hiatal defect in the crura. This repair is done by stitching the crura so that the hole at the hiatal canal is smaller. All of this can be done laparoscopically or in an open procedure through the abdomen or the chest. There are multiple variations on the operation, but the most commonly performed is the laparoscopic Nissen fundoplication.

G&H What other therapeutic options are there for these patients?

JP First, these patients need to be thoroughly examined to determine the extent of the hernia, as there can be serious complications if there is an extensive paraesophageal component. If the hernia is large enough, patients will not respond to PPI therapy alone. They will still get significant acid exposure and caustic exposure at the esophagogastric junction and possibly other extraesophageal manifestations.

In these patients, short of surgery, I recommend lifestyle modifications. They should not eat late at night, and they should raise the head of their bed so that gravity can actually help them clear acid during sleep. This is a mechanical defect that can be controlled somewhat by altering how the patient sleeps.

G&H What further research is needed in the study of reflux disorders and hiatal hernia?

JP Future research will focus on the relationship between the lower esophageal sphincter and the crural diaphragm. One interesting idea to explore is that these two mechanisms are not mutually exclusive. They work best when positioned together, and just by separating them, both components are weakened. A group from the Netherlands recently published a study in *Gastroenterology* showing that reflux events, for the most part, occur when the two sphincters are actually separate.

There should also be more research into other surgical procedures. Although the Nissen fundoplication is a good operation, it is associated with a number of complications. We need to better understand what benefit is accrued by the fundoplication and what we can expect from simply repairing the hernia and repositioning the lower esophageal sphincter back into the abdominal cavity. I would also like to see the development of an antireflux motility agent that increases peristalsis and strengthens the lower esophageal sphincter without surgical intervention. There are also agents that are being developed to block transient lower esophageal sphincter relaxation, similar to baclofen.

Suggested Reading

Pandolfino JE, El-Serag HB, Zhang Q, et al. Obesity: a challenge to esophagogastric junction integrity. *Gastroenterology*. 2006;130:639-649.

Stacher G, Lenglinger J, Eisler M, et al. Esophageal acid exposure in upright and recumbent postures: roles of lower esophageal sphincter, esophageal contractile and transport function, hiatal hernia, age, sex, and body mass. *Dig Dis Sci.* 2006;51:1896-1903.

Bredenoord AJ, Weusten BL, Timmer R, et al. Intermittent spatial separation of diaphragm and lower esophageal sphincter favors acidic and weakly acidic reflux. *Gastroenterology*. 2006;130:334-340.

Harding SM. The potential role of gastroesophageal reflux in asthma. *Minerva Gastroenterol Dietol*. 2001;47:75-83.

Kahrilas PJ. The role of hiatus hernia in GERD. *Yale J Biol Med*. 1999;72:101-111.